RESEARCH ARTICLE

Arbovirus vectors of epidemiological concern in the Americas: A scoping review of entomological studies on Zika, dengue and chikungunya virus vectors

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Abstract

Background

Three arthropod-borne viruses (arboviruses) causing human disease have been the focus of a large number of studies in the Americas since 2013 due to their global spread and epidemiological impacts: Zika, dengue, and chikungunya viruses. A large proportion of infections by these viruses are asymptomatic. However, all three viruses are associated with moderate to severe health consequences in a small proportion of cases. Two mosquito species, *Aedes aegypti* and *Aedes albopictus*, are among the world's most prominent arboviral vectors, and are known vectors for all three viruses in the Americas.

Objectives

This review summarizes the state of the entomological literature surrounding the mosquito vectors of Zika, dengue and chikungunya viruses and factors affecting virus transmission. The rationale of the review was to identify and characterize entomological studies that have been conducted in the Americas since the introduction of chikungunya virus in 2013, encompassing a period of arbovirus co-circulation, and guide future research based on identified knowledge gaps.

Methods

The preliminary search for this review was conducted on PubMed (National Library of Health, Bethesda, MD, United States). The search included the terms 'zika' OR 'dengue'

OR 'chikungunya' AND 'vector' OR 'Aedes aegypti' OR 'Aedes albopictus'. The search was conducted on March 1ˢᵗ of 2018, and included all studies since January 1ˢᵗ of 2013.

Results

A total of 96 studies were included in the scoping review after initial screening and subsequent exclusion of out-of-scope studies, secondary data publications, and studies unavailable in English language.

Key findings

We observed a steady increase in number of publications, from 2013 to 2018, with half of all studies published from January 2017 to March 2018. Interestingly, information on Zika virus vector species composition was abundant, but sparse on Zika virus transmission dynamics. Few studies examined natural infection rates of Zika virus, vertical transmission, or co-infection with other viruses. This is in contrast to the wealth of research available on natural infection and co-infection for dengue and chikungunya viruses, although vertical transmission research was sparse for all three viruses.

Introduction

Arboviruses, or arthropod-borne viruses, comprise a diverse group of viruses mostly transmitted by mosquitoes and ticks, including globally spreading viruses causing human disease, such as Zika, dengue, and chikungunya viruses. The term arbovirus does not encompass a taxonomically distinct group, but these viruses have similar life-history and transmission patterns that make information gleaned from one virus potentially useful to the understanding, and therefore prevention and control, of the others.

Since its identification in Uganda in 1947, Zika virus (*Flavivirus*, *Flaviviridae*) has been, until recently, confined only to Africa and Asia [1]. The virus ultimately reached the Americas in late 2014, resulting in the declaration of a Public Health Emergency of International Concern by the World Health Organization [2]. To date, 86 countries have reported evidence of mosquito-transmitted Zika virus infection. [3] Brazil currently faces the greatest burden of Zika virus infections [4]. Dengue fever, caused by four different serotypes of dengue virus (*Flavivirus*, *Flaviviridae*) is the most common arboviral disease that affects humans– 50 million people contract it each year, and an estimated 22,000 die from severe dengue [5]. Dengue is hyperendemic in the Americas, with cyclic epidemics occurring every three to five years [6]. Chikungunya virus (*Alphavirus*, *Togoviridae*) was first isolated in Tanzania in 1952 [7]. In the early 2000s, chikungunya virus cases and outbreaks were identified in countries in Africa, Asia, and Europe [7]. In 2013, it emerged in the Americas in Saint-Martin, and within the first year, over a million new cases were reported, spreading to 45 countries in the Latin American and Caribbean region [8].

A large proportion of Zika, dengue, and chikungunya viral infections are asymptomatic [9–11]. However, all three viruses are associated with moderate to severe health consequences in a small proportion of cases, with neonates, young children and/or older age groups at higher risk. Symptoms of Zika viral infection include rash, fever, arthralgia, and conjunctivitis [11]. More importantly, since its initial emergence in the Americas, Zika virus has been confirmed as a cause of congenital abnormalities (in infants born to women infected with Zika virus

during pregnancy) and as a trigger of Guillain-Barré Syndrome [12]. Symptoms of dengue viral infection include rash, fever, arthralgia, and nausea. Some of the more severe symptoms of dengue viral infection may include deadly hemorrhage and plasma leak [9]. Symptoms of chikungunya viral infection include rash, fever, and arthralgia that may persist for an extended duration [7].

Two mosquito species, *Aedes aegypti* and *Aedes albopictus*, are among the world's most prominent arboviral vectors. *Ae. aegypti* originated in sub-Saharan Africa as a sylvatic species and was introduced to the Americas via ships soon after European arrival in the 1400s [13]. The species became domesticated and is now endemic to the Americas and the Asia-Pacific. The range of *Ae. albopictus* was restricted to Asia until the latter part of the 20th century. It is thought to have been introduced to the Western hemisphere through a shipment of used tires in 1985 and has expanded its territory to over 40% of the world's landmass over the course of the past 30 years [14–16].

This review summarizes the state of the literature surrounding the vectors of Zika, dengue and chikungunya viruses and factors affecting virus transmission in the Americas, with a focus on public health implications. Waddell et al. [17] conducted a comprehensive scoping review of the Zika virus literature in 2016. However, the authors identified a limited scope of literature on vector studies, and none specifically looked at vector populations of the Americas, highlighting the need for a scoping review focusing on this area given its relevance in understanding arboviral disease risk in the region. This scoping review aims to identify and characterize the literature pertaining to mosquito species vector competence and aspects of virus transmission dynamics in the Americas since the introduction of chikungunya in 2013. This timeframe includes the introduction of Zika virus and the ongoing co-circulation of three globally spreading arboviruses, namely Zika, dengue and chikungunya viruses.

Methods

This study's search strategy and data extraction protocol were developed *a priori*. The list of definitions for each search term and the data characterization and utility form are available upon request. The review was conducted using PRISMA guidelines for scoping reviews [18]. See S2 Table for this scoping review's checklist. The preliminary search for this review was conducted on PubMed (National Library of Health, Bethesda, MD, United States). The search included the terms 'zika' OR 'dengue' OR 'chikungunya' AND 'vector' OR 'Aedes aegypti' OR 'Aedes albopictus'. The search was conducted on March 1st of 2018, and included all studies since January 1st of 2013. We chose the year 2013 as a start date for our search to reflect the timing of chikungunya virus spread to the Americas, followed in 2014 by Zika virus. These years are thus characterized by co-circulation of multiple globally spreading arboviruses in the region. Upon selection of potentially relevant articles, studies were characterized according to main characteristics including study setting, virus of interest, study design, methods of mosquito collection and analysis, vector species discussed, and main findings. Zotero (Center for History and New Media, George Mason University, United States) was initially used for title and abstract screening. All studies were subsequently transferred to Excel (Microsoft Corporation, Redmond, WA, United States) for data characterization and extraction. Two independent reviewers completed each step of the review following the broad initial screening, which was conducted by one reviewer.

Articles were selected if they were related to vector species composition and/or virus transmission dynamics, if they were related to Zika, dengue and/or chikungunya arboviruses, and if they were related to the ongoing virus circulation in the Americas. Other inclusion criteria included availability of an English language version and investigation of primary data. Studies

that specifically examined the impacts of vector control measures, or studies that were unrelated to vector-borne aspects of disease, vector competence or entomological measures, were excluded due to the degree of scope expansion that would be caused by their inclusion.

Results

Descriptive statistics of scoping review

The search yielded 6267 results. All records were screened, and 5919 were not deemed relevant based on title and abstract content. A total of 348 screened full-text studies were examined for eligibility, and ultimately 96 studies were included in the scoping review (Fig 1; S1 Table). The vast majority of studies were performed exclusively in the field, in the laboratory, or using a modelling framework, and most studies were conducted exclusively on *Ae. aegypti* (Table 1). Studies focusing exclusively on dengue virus were the most numerous, followed by studies focusing exclusively on Zika virus, while studies focusing on chikungunya virus or on a combination of arboviruses were the least numerous (Table 1). Studies on virus transmission dynamics were the most numerous, while studies on aspects of both vector species composition and virus transmission dynamics were the least numerous (Table 1). The average monthly number of studies hovered between 0 and 2 from 2013 to 2016, then increased to 3 or more in 2017 and 2018 (Fig 2), closely reflecting the introductions of chikungunya and Zika viruses in the Americas and subsequent epidemics, respectively.

Vector species composition

Zika virus. There is extensive evidence that *Ae. aegypti* mosquitoes are able to transmit Zika virus in both the laboratory [19–29] and in the field [30–32]. *Ae. albopictus* mosquitoes were also able to transmit Zika virus in experimental studies [22,23], but studies in which both *Ae. aegypti* and *Ae. albopictus* were captured found no Zika virus-infected *Ae. albopictus* [31,32]. Gendernalik et al. [33] and O'Donnell et al. [25] report that *Ae. vexans* mosquitoes are also experimentally competent vectors of Zika virus, but no studies indicated natural *Ae. vexans* infection with Zika virus. *Cx. quinquefasciatus* has been identified by predictive models as a potential vector for Zika virus [34], as have *Sabethes* and *Haemagogus spp*. [35]. Seven studies found that *Cx. quinquefasciatus* mosquitoes were refractory to Zika virus when exposed to infectious blood meals [29,36–42]. Ferreira-de-Brito et al. [31] reported that no *Cx. quinquefasciatus* captured in Brazil were positive for Zika virus. In contrast, Guedes et al. [43] detected Zika virus in the midgut, salivary glands and saliva of artificially fed *Cx. quinquefasciatus* captured in Brazil, using RT-PCR and transmission electron microscopy. The same study also reported Zika virus isolated from two field-caught *Cx. quinquefasciatus* in Brazil.

Dengue virus. *Ae. albopictus* [44–47] and *Ae. aegypti* [27,45,46,48–50] are both experimentally competent to transmit dengue virus. Infection by the virus is observed in field populations of *Ae. albopictus* [51–54], *Ae. aegypti* [51,52,54–62] and *Cx. quinquefasciatus* [56], although the latter was not identified as a competent vector species experimentally.

Chikungunya virus. *Ae. aegypti* [46,63–68], *Ae. albopictus* [46,64,66–69], *Aedes terrens* [70], and *Haemagogus leucocelaenus* [70] are all experimentally competent to transmit chikungunya virus. Chikungunya virus transmission in *Ae. aegypti* has also been observed in the field [30,59,71,72].

Virus transmission dynamics

Vector competence factors. Four studies measured the effect of temperature on vector competence [47,63,64,73]. Adelman et al. [63] found that under silenced RNAi conditions, *Ae.*
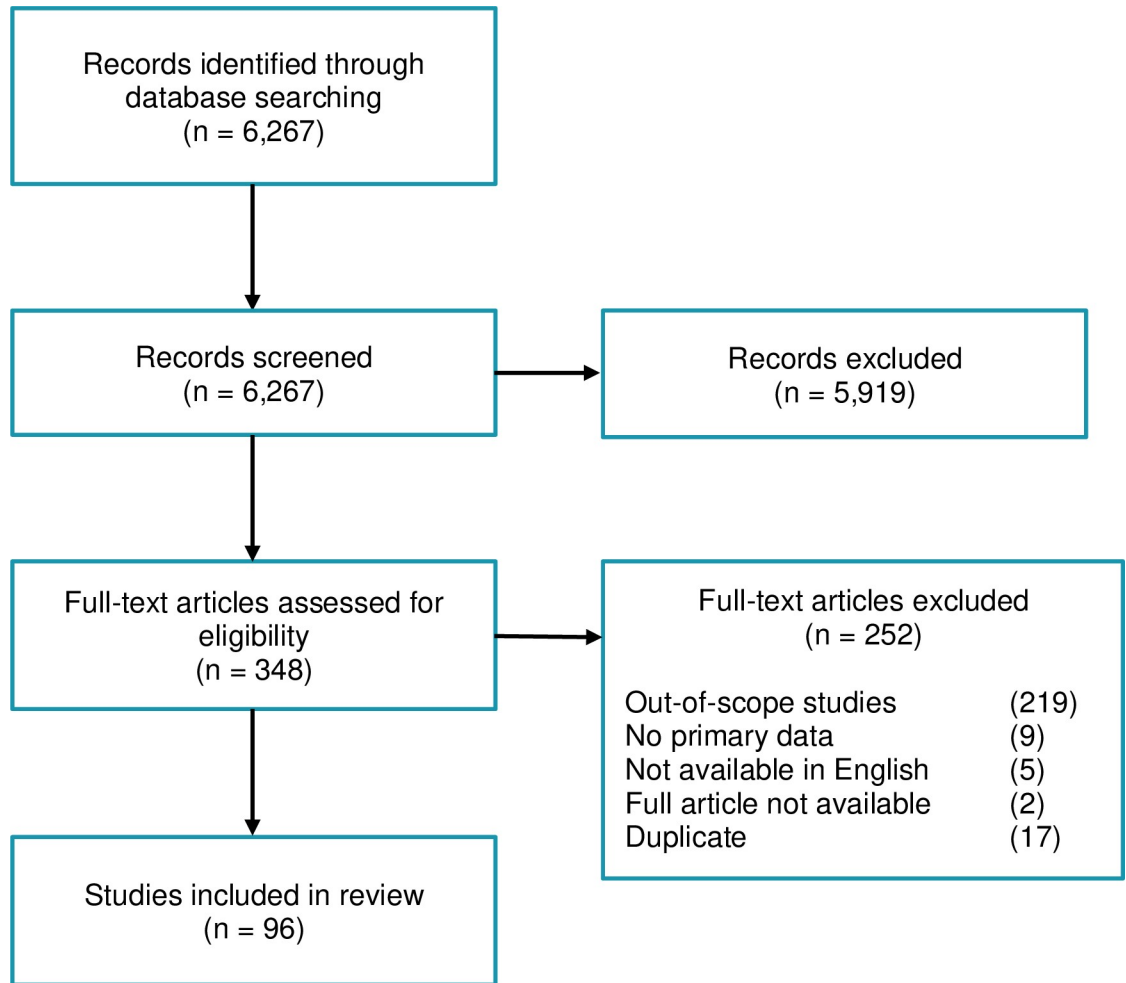
Fig 1. Summary of screening and exclusion steps of this scoping review's methodology, and resulting number of publications after each step.

aegypti were more predisposed to chikungunya infection at lower temperatures. Alto et al. [64] found that larger fluctuations in diurnal temperature range led to higher rates of chikungunya infection, and Xiao et al. [47] found that maximum dengue infection rates occurred at 31°C. Mordecai et al. [73] modelled *Ae. aegypti* and *Ae. albopictus* transmission in the Americas and found that mean temperature data accurately reflected Zika, chikungunya and dengue human case data. Transmission was found to occur between 18 and 34°C and maximal transmission was observed between 26–29°C, with less certainty surrounding the critical thermal minimum than the critical thermal maximum [73]. *Ae. albopictus* was found to perform better in cooler temperatures [73]. Buckner et al. [45] found that the interaction of low temperature and low food availability increased *Ae. aegypti* and *Ae. albopictus* susceptibility to DENV-1 serotype infection.

Three studies examined the effects of larval competition on dengue vector competence [44,45,74]. Bara et al. [44] found that *Ae. albopictus* larval competition resulted in significantly longer development times, lower emergence rates, and smaller adults, but did not significantly affect the extrinsic incubation period of DENV-2 virus. Kang et al. [74] found that larval-stage crowding and nutritional limitation led to lower survival rates until pupation, lower blood

Table 1. Number of publications included in the scoping review, for each review section, study design, and arbovirus and mosquito vector species of interest.

Theme	Category	Number of publications
Section	Vector Species Composition	29
	Virus Transmission Dynamics	42
	Both sections	25
Study design	Field	16
	Laboratory	40
	Modelling	27
	Field and Laboratory	9
	Field and Modelling	3
	Laboratory and Modelling	1
Virus of interest	Zika	30
	Dengue	45
	Chikungunya	10
	Multiple	11
Mosquito species of interest	*Ae. aegypti*	52
	Ae. albopictus	6
	Cx. quinquefasciatus	3
	Ae. aegypti and *Ae. albopictus*	19
	Ae. aegypti and *Cx. quinquefasciatus*	1
	Ae. albopictus and *Cx. quinquefasciatus*	0
	Ae. aegypti, *Ae. albopictus* and *Cx. quinquefasciatus*	1
	Others	12
	None specifically	2

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feeding success, slower development, smaller adult body size, and lower susceptibility to DENV-2 infection. Four studies examined a variety of blood meal characteristics on arboviral infection rate [23,24,49,75]. Fresh Zika-infected blood meal was associated with significantly higher infection rates than frozen Zika-infected blood meal [23]. Similarly, Zika-infected whole blood meal was associated with significantly higher infection rates than Zika-infected protein meal [24]. Hill et al. [49] studied the impact of antibiotics on dengue infection rate and mosquito fertility, and found no significant association in *Ae. aegypti*. Mosquitoes exposed to DENV-2 were more likely to re-feed than those that were unexposed [75]. Sylvestre et al. [76] studied the impact of DENV-2 infection on *Ae. aegypti* life history traits, and found that it significantly affected feeding behaviour, survival, fecundity, and oviposition success.

Vector infection rate. Two studies conducted in Brazil exclusively examined infection rates by Zika virus in wild mosquito populations (Table 2). Ferreira-de-Brito et al. [31] reported three Zika-infected pools of *Ae. aegypti*, but no Zika-infected *Cx. quinquefasciatus* or *Ae. albopictus* pool [31], out of 468 tested pools among the three species. Ayllón et al. [32] tested 406 *Ae. aegypti* and 11 *Ae. albopictus* field-collected individuals, and found three Zika-infected *Ae. aegypti* individuals.

Six studies reported exclusively on dengue infection rates in wild mosquito populations (Table 2). Cecílio et al. [77] observed four positive pools, out of 54 tested, among *Aedes* mosquitoes collected in two regions of Brazil over the course of 17 months, through the installation of ovitraps in public schools. Cruz et al. [57] detected eight positive pools, out of 50 *Ae. aegypti* pools, collected in Mato Grosso, Brazil. Martínez et al. [62] reported two positive pools, out of 226 *Ae. aegypti* pools, collected in Mexico. Claderón-Arguedas et al. [78] reported nine positive pools, out of 35 *Ae. albopictus* pools, collected in Costa Rica. Pérez-Pérez et al. [54] reported
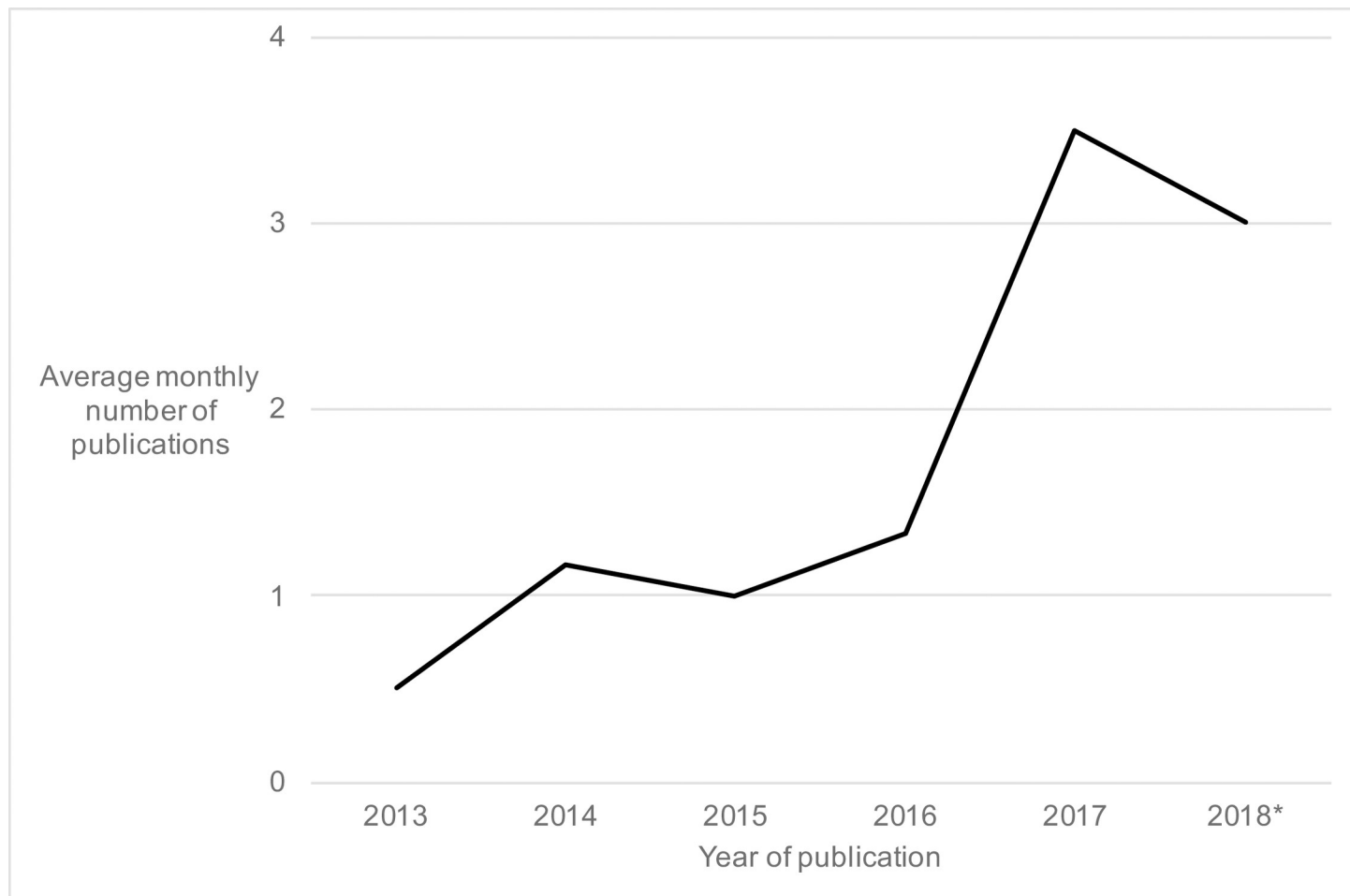
Fig 2. Average monthly number of publications included in the scoping review, for each year since 2013, out of a total of 96. *Year-to-date on March 1st 2018.

Table 2. List of studies that report a proportion of positive mosquito pools for any or a combination of Zika, dengue and chikungunya viruses, along with information on authors, year and country of location of the study, and mosquito species of interest.

Authors	Year	Location	Mosquito species	Pools tested	Zika rate (%)	Dengue rate (%)	Chikungunya rate (%)
Ferreira-de-Brito et al.	2016	Brazil	*Aedes* sp. and *Cx. quinquefasciatus*	468	0.64	ø	ø
Ayllón et al.	2017	Brazil	*Ae. aegypti* and *Ae. albopictus*	178	1.12	ø	ø
Martínez et al.	2014	Mexico	*Ae. aegypti*	226	ø	0.88	ø
Calderón-Arguedas et al.	2015	Costa Rica	*Ae. albopictus*	35	ø	25.71	ø
Cecílio et al.	2015	Brazil	*Aedes* sp.	54	ø	7.41	ø
Cruz et al.	2015	Brazil	*Ae. aegypti*	50	ø	16.00	ø
Pérez-Castro et al.	2016	Colombia	*Ae. aegypti*	34	ø	61.76	ø
Pérez-Pérez et al.	2017	Colombia	*Ae. aegypti* and *Ae. albopictus*	407	ø	32.43	ø
Díaz-González et al.	2015	Mexico	*Ae. aegypti*	557	ø	ø	3.23
Cevallos et al.	2018	Ecuador	*Ae. aegypti*	22	14.29	ø	12.50
Dzul-Manzanilla et al.	2015	Mexico	*Ae. aegypti*	284	ø	9.51	3.17
Cigarroa-Toledo et al.	2016	Mexico	*Ae. aegypti*	27–237*	ø	0.00	0.84–7.40*
Farraudière et al.	2017	Martinique	*Ae. aegypti*	414	ø	1.21	2.66

*Total number of pools tested is not stated, but number of sampled mosquitoes, and maximum number of mosquitoes per pool, are stated.

132 positive pools, out of 407 tested, collected in Colombia. One of the positive pools was *Ae. albopictus*, and the remainder were *Ae. aegypti*. Pérez-Castro et al. [79] reported 21 positive pools, out of 34 tested, in *Ae. aegypti* in Colombia.

A study measured the naturally-occurring prevalence of chikungunya virus in wild mosquito populations (Table 2). Díaz-González et al. [72] reported 18 *Ae. aegypti* positive pools in Mexico, out of 557 tested. A study reported on the prevalence of both chikungunya and Zika viruses among *Ae. aegypti* in Ecuador (Table 2) [30]. Three studies tested both chikungunya and dengue viruses in wild mosquito populations (Table 2). Chikungunya, but not dengue, was detected in *Ae. aegypti* in Mexico by Cigarroa-Toledo et al. [71], although both chikungunya and dengue viruses were isolated in Mexico in *Ae. aegypti* by Dzul-Manzanilla et al. [59], and in Martinique by Farraudière et al. [61].

Vertical transmission. Three studies reported on vertical transmission of dengue virus [58,60,80], and one [81] reported on the vertical transmission of Zika virus. Buckner et al. [80] found a vertical transmission rate of DENV-1 of 11.11% in *Ae. albopictus* and of 8.33% in *Ae. aegypti*. Da Costa et al. [58] observed dengue infection rates among third and fourth instar *Ae. aegypti* between 1.14% and 2.41% in Brazilian municipalities, and Espinosa et al. [60] observed one DENV-3 positive male *Ae. aegypti* pool, collected in Argentina. Thangamani et al. [81] experimentally injected mosquitoes with Zika virus and observed Zika virus infection in *Ae. aegypti* offspring, but not *Ae. albopictus*. Six filial *Ae. aegypti* pools out of 69 tested were found positive for Zika virus [81].

Transmission risk modelling. Seven studies modelled transmission dynamics for Zika virus [40,82–87]. Lourenço et al. [40] used vectorial capacity as a means of prediction, Marini et al. [82] and Majumder et al. [83] used vector abundance and human case data, and Villela et al. [84] and Ospina et al. [85] used disease notification and natural history. Rojas et al. [86] found attack rates in Girardot and San Andres, Colombia to be highest among females, aged 20–49. Fitzgibbon et al. [87] report that early host and vector heterogeneity significantly affect final epidemic size.

Eleven studies modelled dengue transmission dynamics [88–99]. Lee et al. [95] constructed a predictive model that accurately foresaw 75% of dengue outbreaks in Colombia. Reiner et al. [88] reported that social proximity drives fine-scale heterogeneity in dengue transmission rates based on data from Peru. Three studies reported that meteorological variables including temperature and humidity are important determinants of transmission dynamics [89,90,92,93], and one study found that transovarial transmission plays an important role in transmission dynamics depending on basic reproductive number [91]. Liu-Helmersson et al. [96] predicted an increase in diurnal temperature range and increased dengue epidemic potential under climate changes in cold, temperate and extremely hot climates where mean temperatures are far from 29˚C. Velasques-Castro et al. [97] studied *Ae. aegypti* dynamics in relation to host spatial heterogeneity and generated a dengue infection risk map, based on host dynamics. Taber et al. [98] modelled the colonization of Pennsylvania by *Ae. albopictus* together with corresponding risk of dengue.

One study estimated chikungunya transmission risk according to temperature threshold for breeding and adult mosquitoes in Argentina [99]. The authors suggest that temperatures conducive to *Ae. aegypti* breeding and transmission are present during September and April in northeastern Argentina, and in January in southern Argentina. A study compared endemic and transient chikungunya and dengue transmission dynamics, and the role of virus evolution [100]. They found that reducing biting rate and vector-to-susceptible-host ratio were the most effective at reducing basic reproductive number. A study modelled transmission risk of Zika, dengue and chikungunya and found temperature data to match well with human case data [73].

Strain infectivity and co-infection. Six studies examined the infectivity of different dengue viral strains, and the impact of co-infection [50,74,101–104]. Muturi et al. [50] found that infection with DENV-4 rendered *Ae. aegypti* significantly less susceptible to secondary infection with DENV-2. Kang et al. [74] modelled interactions between dengue viral serotypes. Quiner et al. [101] studied the infectivity of different isolates of DENV-2, and found NI-2B to have a replicative advantage over NI-1 until 12 days following infection, after which the advantage had dissipated. Quintero-Gil et al. [102] found that the DENV-2 serotype performed with a thousand-fold greater efficiency than the DENV-3 serotype, upon co-infection. In parallel, Serrato-Salas et al. [103] found that *Ae. aegypti* were significantly less susceptible to secondary dengue infection, after having been challenged with an inactive version of the virus. Vazeille et al. [104] found that DENV-4 outperformed DENV-1 in *Ae. aegypti* upon co-infection. Nuckols et al. [46] artificially infected *Ae. aegypti* and *Ae. albopictus* with chikungunya and DENV-2 simultaneously, separately, and in reverse order. Simultaneous dissemination was detected in all groups upon co-infection, and co-transmission occurred at low rates [46]. Rückert et al. [27] found that the co-infection of *Ae. aegypti* with Zika, chikungunya and dengue viruses minimally affected vector competence, and that vectors were able to transmit each viral pair, as well as three viruses simultaneously. Alto et al. [69] found *Ae. aegypti* and *Ae. albopictus* to be susceptible to Indian Ocean and Asian chikungunya virus genotypes.

Human disease risk. Five articles studied correlations between entomological measures and risk of human dengue infection [105–109]. One study conducted in Peru found that *Ae. aegypti* density was not associated with an increased risk of seroconversion [105]. One study in Acre, Brazil found that *Ae. aegypti* density and risk of dengue increased with tourism and case importation [106]. A study in Mexico City found a positive correlation between dengue incidence and *Ae. aegypti* indoor abundance, as well as monthly average temperature and rainfall [107]. Another study conducted in Peru found that an individual's likelihood of being bitten in the home was directly proportional to time spent in the home, and body surface area. They did not find age or gender to be significant predictors [108]. Oliveira et al. [109] reported the circulation of four dengue serotypes in Brazil introduced between 2001 and 2012 (DENV-1, DENV-2, DENV-3, DENV-4) and reported an increase in dengue infection in Brazil during that time period, i.e. 587 cases/100 000 in 2001 to 1561 cases/100 000 in 2012. Monaghan et al. [110] predicted the seasonal abundance of *Ae. aegypti* in the United States using meterorologically driven models as a means of estimating arboviral infection risk [110]. All 50 included cities were found to be suitable during the summer months (July to September), while only cities in Florida and Texas were found to have *Ae. aegypti* abundance potential during the winter months (December to March). Lo and Park [111] found that regions of Brazil with elevated temperature and precipitation were more conducive to *Ae. aegypti* presence and Zika virus cases. Da Cruz Ferreira et al. [112] found that dengue occurrence increased by 25% when the average number of mosquitoes caught by traps increased by 0.1 per week. Stewart-Ibarra and Lowe [113] assessed the effect of climatic and entomological variables on intra-annual variability in dengue incidence in Southern Ecuador. Da Rocha Taranto et al. [114] examined the relationship between vector collection, species composition, hatching rates, and population density on dengue incidence. Hatching rate was found to be affected by population density and climate, and presence of vectors was associated with dengue incidence [114]. Ernst et al. [94] found no correlation between *Ae. aegypti* density and human age structure between two cities with different dengue transmission dynamics.

Discussion

Our scoping review included studies focused on vector species composition and arbovirus transmission dynamics of Zika, dengue and/or chikungunya in the Americas. We observed a steady

increase in number of publications, from 2013 to 2018, with half of all studies published from January 2017 to March 2018. Sightly less than half of all studies included in this review were specifically pertaining to virus transmission dynamics. Around a third of all studies addressed vector species composition. The remainder treated aspects of both sections. Most studies focused on *Aedes aegypti* as the vector species of interest, had an exclusively laboratory-based or modelling-based study framework, and focused exclusively on either Zika or dengue. One limitation of our study is the use of a single search engine, PubMed, which may have reduced the number of included publications in our scoping review. However, given the focus of our scoping review, we believe this search engine should have captured almost all, if not all, relevant studies.

To determine vector competence, a species must be able to acquire, maintain, and transmit a pathogen, which is assessed through experimental infection studies. However, these studies are heterogeneous in both the mosquito populations and virus strains used, as well as methods measuring potential to transmit [115]. The detection of viral particles in wild-caught mosquitoes does not signify vector competence on its own, but it lends support to evidence from laboratory studies, when coupled with the observation of human host-feeding behaviour. Field studies are also important to assess the relative importance of competent vector species in disease maintenance and/or transmission. Vector competence for Zika virus has been well established for *Ae. aegypti* [19–32] and *Ae. albopictus* [22,23], but there is a growing consensus that *Cx. quinquefasciatus* is not a competent Zika virus vector, and no consensus has been reached regarding the competence of *Ae vexans*. A number of studies report that *Cx. quinquefasciatus* is refractory to Zika virus [29,36–39,41,116]. While Zika virus has been detected in a small number of field-caught *Cx. quinquefasciatus* in Brazil [42], this does not necessarily indicate their ability to transmit the virus. Interestingly, information on Zika virus vector species composition was abundant, but sparse on Zika virus transmission dynamics. Few studies examined natural infection rates of Zika virus [31,32], vertical transmission [81], or co-infection with other viruses [27]. This is in contrast to the wealth of research available on natural infection and co-infection for dengue and chikungunya viruses, although vertical transmission research was sparse for all three viruses [46,50,58,77,80,101,102].

Based on the internationally recognized urgency of Zika virus infection as a public health concern, and potential increase in the importance of this and other emerging arboviruses in the future, further research on Zika virus transmission dynamics is of pressing need. Also, given the ongoing co-circulation of these three globally spreading arboviruses in the Americas, and the resulting complexity of their transmission dynamics, more integrative studies are needed that investigate a combination of Zika, dengue and chikungunya viruses and use a variety of approaches to answer questions relating to the risk posed by these arboviruses.

Supporting information

S1 Table. List of full-text articles included in the review. Information on first author's last name, year of publication, title, journal, review section, study design, and arbovirus and mosquito vector species of interest are given for each full-text article.
(XLSX)

S2 Table. PRISMA-ScR checklist. Checklist stating location of each element of the scoping review, as implemented by Tricco et al. [18].
(PDF)

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References

1. Dick GWA, Kitchen SF, Haddow AJ. Zika virus. I. Isolations and serological specificity. Trans R Soc Trop Med Hyg. 1952; 46: 509–520. https://doi.org/10.1016/0035-9203(52)90042-4 PMID: 12995440

2. Weaver SC, Costa F, Garcia-Blanco MA, Ko AI, Ribeiro GS, Saade G, et al. Zika virus: History, emergence, biology, and prospects for control. Antiviral Res. 2016; 130: 69–80. https://doi.org/10.1016/j.antiviral.2016.03.010 PMID: 26996139

3. World Health Organization. Zika virus. 2018. Available: https://www.who.int/news-room/fact-sheets/detail/zika-virus

4. Hills SL, Fischer M, Petersen LR. Epidemiology of Zika virus infection. J Infect Dis. 2017; 216: S868–S874. https://doi.org/10.1093/infdis/jix434 PMID: 29267914

5. Kamgang B, Yougang AP, Tchoupo M, Riveron JM, Wondji C. Temporal distribution and insecticide resistance profile of two major arbovirus vectors Aedes aegypti and Aedes albopictus in Yaoundé, the capital city of Cameroon. Parasit Vectors. 2017; 10: 469. https://doi.org/10.1186/s13071-017-2408-x PMID: 29017606

6. Murray NEA, Quam MB, Wilder-Smith A. Epidemiology of dengue: past, present and future prospects. Clin Epidemiol. 2013; 5: 299–309. https://doi.org/10.2147/CLEP.S34440 PMID: 23990732

7. Zeller H, Van Bortel W, Sudre B. Chikungunya: its history in Africa and Asia and its spread to new regions in 2013–2014. J Infect Dis. 2016; 214: S436–S440. https://doi.org/10.1093/infdis/jiw391 PMID: 27920169

8. Yactayo S, Staples JE, Millot V, Cibrelus L, Ramon-Pardo P. Epidemiology of chikungunya in the Americas. J Infect Dis. 2016; 214: S441–S445. https://doi.org/10.1093/infdis/jiw390 PMID: 27920170

9. Centre for Disease Control. Clinical Guidance | Dengue | CDC. 2019. Available: https://www.cdc.gov/dengue/clinicallab/clinical.html

10. Nakkhara P, Chongsuvivatwong V, Thammapalo S. Risk factors for symptomatic and asymptomatic chikungunya infection. Trans R Soc Trop Med Hyg. 2013; 107: 789–796. https://doi.org/10.1093/trstmh/trt083 PMID: 24052594

11. Haby MM, Pinart M, Elias V, Reveiz L. Prevalence of asymptomatic Zika virus infection: a systematic review. Bull World Health Organ. 2018; 96: 402–413D. https://doi.org/10.2471/BLT.17.201541 PMID: 29904223

12. Krauer F, Riesen M, Reveiz L, Oladapo OT, Martínez-Vega R, Porgo TV, et al. Zika virus infection as a cause of congenital brain abnormalities and Guillain-Barré syndrome: Systematic Review. PLoS Med. 2017; 14: e1002203. https://doi.org/10.1371/journal.pmed.1002203 PMID: 28045901

13. Powell JR, Tabachnick WJ, Powell JR, Tabachnick WJ. History of domestication and spread of *Aedes aegypti*—a review. Mem Inst Oswaldo Cruz. 2013; 108: 11–17. https://doi.org/10.1590/0074-0276130395 PMID: 24473798

14. Wagman J, Grieco JP, King R, Briceño I, Bautista K, Polanco J, et al. First record and demonstration of a southward expansion of *Aedes albopictus* into Orange Walk Town, Belize, Central America. J Am Mosq Control Assoc. 2013; 29: 380–382. https://doi.org/10.2987/13-6364.1 PMID: 24551972

15. Kraemer MUG, Reiner RC, Brady OJ, Messina JP, Gilbert M, Pigott DM, et al. Past and future spread of the arbovirus vectors *Aedes aegypti* and *Aedes albopictus.* Nat Microbiol. 2019; 4: 854–863. https://doi.org/10.1038/s41564-019-0376-y PMID: 30833735

16. Miller MJ, Loaiza JR. Geographic expansion of the invasive mosquito *Aedes albopictus* across Panama—implications for control of dengue and chikungunya viruses. PLoS Negl Trop Dis. 2015; 9: e0003383. https://doi.org/10.1371/journal.pntd.0003383 PMID: 25569303

17. Waddell LA, Greig JD. Scoping review of the Zika virus literature. PloS One. 2016; 11: e0156376. https://doi.org/10.1371/journal.pone.0156376 PMID: 27244249

18. Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Levac D, et al. PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. Ann Intern Med. 2018; 169: 467. https://doi.org/10.7326/M18-0850 PMID: 30178033

19. Bolling BG, Vasilakis N, Guzman H, Widen SG, Wood TG, Popov VL, et al. Insect-specific viruses detected in laboratory mosquito colonies and their potential implications for experiments evaluating arbovirus vector competence. Am J Trop Med Hyg. 2015; 92: 422–428. https://doi.org/10.4269/ajtmh.14-0330 PMID: 25510714

20. Azar SR, Roundy CM, Rossi SL, Huang JH, Leal G, Yun R, et al. Differential vector competency of *Aedes albopictus* populations from the Americas for Zika virus. Am J Trop Med Hyg. 2017; 97: 330–339. https://doi.org/10.4269/ajtmh.16-0969 PMID: 28829735

21. Costa-da-Silva AL, Ioshino RS, Araújo HRC de, Kojin BB, Zanotto PM de A, Oliveira DBL, et al. Correction: laboratory strains of *Aedes aegypti* are competent to Brazilian Zika virus. PloS One. 2017; 12: e0174081. https://doi.org/10.1371/journal.pone.0174081 PMID: 28288192

22. Chouin-Carneiro T, Vega-Rua A, Vazeille M, Yebakima A, Girod R, Goindin D, et al. Differential susceptibilities of Aedes aegypti and *Aedes albopictus* from the Americas to Zika virus. PLoS Negl Trop Dis. 2016; 10: e0004543. https://doi.org/10.1371/journal.pntd.0004543 PMID: 26938868

23. Ciota AT, Bialosuknia SM, Zink SD, Brecher M, Ehrbar DJ, Morrissette MN, et al. Effects of Zika virus strain and *Aedes Mosquito* species on vector competence. Emerg Infect Dis. 2017; 23: 1110–1117. https://doi.org/10.3201/eid2307.161633 PMID: 28430564

24. Huang Y-JS, Lyons AC, Hsu W-W, Park SL, Higgs S, Vanlandingham DL. Differential outcomes of Zika virus infection in Aedes aegypti orally challenged with infectious blood meals and infectious protein meals. PloS One. 2017; 12: e0182386. https://doi.org/10.1371/journal.pone.0182386 PMID: 28796799

25. O'Donnell KL, Bixby MA, Morin KJ, Bradley DS, Vaughan JA. Potential of a northern population of *Aedes vexans* (Diptera: Culicidae) to transmit Zika virus. J Med Entomol. 2017; 54: 1354–1359. https://doi.org/10.1093/jme/tjx087 PMID: 28499036

26. Roundy CM, Azar SR, Rossi SL, Huang JH, Leal G, Yun R, et al. Variation in *Aedes aegypti* mosquito competence for Zika virus transmission. Emerg Infect Dis. 2017; 23: 625–632. https://doi.org/10.3201/eid2304.161484 PMID: 28287375

27. Rückert C, Weger-Lucarelli J, Garcia-Luna SM, Young MC, Byas AD, Murrieta RA, et al. Impact of simultaneous exposure to arboviruses on infection and transmission by *Aedes aegypti* mosquitoes. Nat Commun. 2017; 8: 15412. https://doi.org/10.1038/ncomms15412 PMID: 28524874

28. Secundino NFC, Chaves BA, Orfano AS, Silveira KRD, Rodrigues NB, Campolina TB, et al. Zika virus transmission to mouse ear by mosquito bite: a laboratory model that replicates the natural transmission process. Parasit Vectors. 2017; 10: 346. https://doi.org/10.1186/s13071-017-2286-2 PMID: 28728607

29. Weger-Lucarelli J, Rückert C, Chotiwan N, Nguyen C, Garcia Luna SM, Fauver JR, et al. Vector competence of American mosquitoes for three strains of Zika virus. PLoS Negl Trop Dis. 2016; 10: e0005101. https://doi.org/10.1371/journal.pntd.0005101 PMID: 27783679

30. Cevallos V, Ponce P, Waggoner JJ, Pinsky BA, Coloma J, Quiroga C, et al. Zika and chikungunya virus detection in naturally infected *Aedes aegypti* in Ecuador. Acta Trop. 2018; 177: 74–80. https://doi.org/10.1016/j.actatropica.2017.09.029 PMID: 28982578

31. Ferreira-de-Brito A, Ribeiro IP, Miranda RM de, Fernandes RS, Campos SS, Silva KAB da, et al. First detection of natural infection of *Aedes aegypti* with Zika virus in Brazil and throughout South America. Mem Inst Oswaldo Cruz. 2016; 111: 655–658. https://doi.org/10.1590/0074-02760160332 PMID: 27706382

32. Ayllón T, Campos R de M, Brasil P, Morone FC, Câmara DCP, Meira GLS, et al. Early evidence for Zika virus circulation among *Aedes aegypti* mosquitoes, Rio de Janeiro, Brazil. Emerg Infect Dis. 2017; 23: 1411–1412. https://doi.org/10.3201/eid2308.162007 PMID: 28628464

33. Gendernalik A, Weger-Lucarelli J, Garcia Luna SM, Fauver JR, Rückert C, Murrieta RA, et al. American *Aedes vexans* mosquitoes are competent vectors of Zika virus. Am J Trop Med Hyg. 2017; 96: 1338–1340. https://doi.org/10.4269/ajtmh.16-0963 PMID: 28719283

34. Evans MV, Dallas TA, Han BA, Murdock CC, Drake JM. Data-driven identification of potential Zika virus vectors. eLife. 2017; 6. https://doi.org/10.7554/eLife.22053 PMID: 28244371

35. Althouse BM, Vasilakis N, Sall AA, Diallo M, Weaver SC, Hanley KA. Potential for Zika virus to establish a sylvatic transmission cycle in the Americas. PLoS Negl Trop Dis. 2016; 10: e0005055. https://doi.org/10.1371/journal.pntd.0005055 PMID: 27977671

36. Fernandes RS, Campos SS, Ribeiro PS, Raphael LM, Bonaldo MC, Lourenço-de-Oliveira R. *Culex quinquefasciatus* from areas with the highest incidence of microcephaly associated with Zika virus infections in the Northeast Region of Brazil are refractory to the virus. Mem Inst Oswaldo Cruz. 2017; 112: 577–579. https://doi.org/10.1590/0074-02760170145 PMID: 28767975

37. Fernandes RS, Campos SS, Ferreira-de-Brito A, Miranda RM de, Barbosa da Silva KA, Castro MG de, et al. *Culex quinquefasciatus* from Rio de Janeiro is not competent to transmit the local Zika virus. PLoS Negl Trop Dis. 2016; 10: e0004993. https://doi.org/10.1371/journal.pntd.0004993 PMID: 27598421

38. Hart CE, Roundy CM, Azar SR, Huang JH, Yun R, Reynolds E, et al. Zika virus vector competency of mosquitoes, Gulf Coast, United States. Emerg Infect Dis. 2017; 23: 559–560. https://doi.org/10.3201/eid2303.161636 PMID: 28005002

39. Kenney JL, Romo H, Duggal NK, Tzeng W-P, Burkhalter KL, Brault AC, et al. Transmission incompetence of *Culex quinquefasciatus* and *Culex pipiens pipiens* from North America for Zika virus. Am J Trop Med Hyg. 2017; 96: 1235–1240. https://doi.org/10.4269/ajtmh.16-0865 PMID: 28500817

40. Lourenço J, Maia de Lima M, Faria NR, Walker A, Kraemer MU, Villabona-Arenas CJ, et al. Epidemiological and ecological determinants of Zika virus transmission in an urban setting. eLife. 2017; 6. https://doi.org/10.7554/eLife.29820 PMID: 28887877

41. Dodson BL, Rasgon JL. Vector competence of *Anopheles* and *Culex* mosquitoes for Zika virus. PeerJ. 2017; 5: e3096. https://doi.org/10.7717/peerj.3096 PMID: 28316896

42. Aliota MT, Peinado SA, Osorio JE, Bartholomay LC. *Culex pipiens* and *Aedes triseriatus* mosquito susceptibility to Zika virus. Emerg Infect Dis. 2016; 22: 1857–1859. https://doi.org/10.3201/eid2210.161082 PMID: 27434194

43. Guedes DR, Paiva MH, Donato MM, Barbosa PP, Krokovsky L, Rocha SWDS, et al. Zika virus replication in the mosquito *Culex quinquefasciatus* in Brazil. Emerg Microbes Infect. 2017; 6: e69. https://doi.org/10.1038/emi.2017.59 PMID: 28790458

44. Bara J, Rapti Z, Cáceres CE, Muturi EJ. Effect of larval competition on extrinsic incubation period and vectorial capacity of *Aedes albopictus* for dengue virus. PloS One. 2015; 10: e0126703. https://doi.org/10.1371/journal.pone.0126703 PMID: 25951173

45. Buckner EA, Alto BW, Lounibos LP. Larval temperature-food effects on adult mosquito infection and vertical transmission of dengue-1 virus. J Med Entomol. 2016; 53: 91–98. https://doi.org/10.1093/jme/tjv145 PMID: 26489999

46. Nuckols JT, Huang Y-JS, Higgs S, Miller AL, Pyles RB, Spratt HM, et al. Evaluation of simultaneous transmission of chikungunya virus and dengue virus type 2 in infected *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae). J Med Entomol. 2015; 52: 447–451. https://doi.org/10.1093/jme/tjv017 PMID: 26334820

47. Xiao F-Z, Zhang Y, Deng Y-Q, He S, Xie H-G, Zhou X-N, et al. The effect of temperature on the extrinsic incubation period and infection rate of dengue virus serotype 2 infection in *Aedes albopictus*. Arch Virol. 2014; 159: 3053–3057. https://doi.org/10.1007/s00705-014-2051-1 PMID: 24990415

48. Lourenço-de-Oliveira R, Rua AV, Vezzani D, Willat G, Vazeille M, Mousson L, et al. *Aedes aegypti* from temperate regions of South America are highly competent to transmit dengue virus. BMC Infect Dis. 2013; 13: 610. https://doi.org/10.1186/1471-2334-13-610 PMID: 24373423

49. Hill CL, Sharma A, Shouche Y, Severson DW. Dynamics of midgut microflora and dengue virus impact on life history traits in *Aedes aegypti*. Acta Trop. 2014; 140: 151–157. https://doi.org/10.1016/j.actatropica.2014.07.015 PMID: 25193134

50. Muturi EJ, Buckner E, Bara J. Superinfection interference between dengue-2 and dengue-4 viruses in *Aedes aegypti* mosquitoes. Trop Med Int Health TM IH. 2017; 22: 399–406. https://doi.org/10.1111/tmi.12846 PMID: 28150899

51. Dos Santos TP, Cruz OG, da Silva KAB, de Castro MG, de Brito AF, Maspero RC, et al. Dengue serotype circulation in natural populations of *Aedes aegypti*. Acta Trop. 2017; 176: 140–143. https://doi.org/10.1016/j.actatropica.2017.07.014 PMID: 28743449

52. Alto BW, Smartt CT, Shin D, bBettinardi D, Malicoate J, Anderson SL, et al. Susceptibility of Florida *Aedes aegypti* and *Aedes albopictus* to dengue viruses from Puerto Rico. J Vector Ecol J Soc Vector Ecol. 2014; 39: 406–413. https://doi.org/10.1111/jvec.12116 PMID: 25424270

53. Gómez-Palacio A, Suaza-Vasco J, Castaño S, Triana O, Uribe S. *Aedes albopictus* (Skuse, 1894) infected with the American-Asian genotype of dengue type 2 virus in Medellín suggests its possible role as vector of dengue fever in Colombia. Biomed Rev Inst Nac Salud. 2017; 37: 135–142.

54. Pérez-Pérez J, Sanabria WH, Restrepo C, Rojo R, Henao E, Triana O, et al. Virological surveillance of *Aedes* (*Stegomyia*) *aegypti* and *Aedes* (*Stegomyia*) *albopictus* as support for decision making for dengue control in Medellín. Biomed Rev Inst Nac Salud. 2017; 37: 155–166.

55. Gonçalves CM, Melo FF, Bezerra JMT, Chaves BA, Silva BM, Silva LD, et al. Distinct variation in vector competence among nine field populations of Aedes aegypti from a Brazilian dengue-endemic risk city. Parasit Vectors. 2014; 7: 320. https://doi.org/10.1186/1756-3305-7-320 PMID: 25015526

56. Serra OP, Cardoso BF, Ribeiro ALM, Santos FAL dos, Slhessarenko RD. Mayaro virus and dengue virus 1 and 4 natural infection in culicids from Cuiabá, state of Mato Grosso, Brazil. Mem Inst Oswaldo Cruz. 2016; 111: 20–29. https://doi.org/10.1590/0074-02760150270 PMID: 26784852

57. Cruz LC de TA da, Serra OP, Leal-Santos FA, Ribeiro ALM, Slhessarenko RD, Santos MA dos. Natural transovarial transmission of dengue virus 4 in *Aedes aegypti* from Cuiabá, State of Mato Grosso, Brazil. Rev Soc Bras Med Trop. 2015; 48: 18–25. https://doi.org/10.1590/0037-8682-0264-2014 PMID: 25860459

58. da Costa CF, Dos Passos RA, Lima JBP, Roque RA, de Souza Sampaio V, Campolina TB, et al. Transovarial transmission of DENV in *Aedes aegypti* in the Amazon basin: a local model of xenomonitoring. Parasit Vectors. 2017; 10: 249. https://doi.org/10.1186/s13071-017-2194-5 PMID: 28526066

59. Dzul-Manzanilla F, Martínez NE, Cruz-Nolasco M, Gutiérrez-Castro C, López-Damián L, Ibarra-López J, et al. Arbovirus surveillance and first report of chikungunya virus in wild populations of *Aedes aegypti* from Guerrero, Mexico. J Am Mosq Control Assoc. 2015; 31: 275–277. https://doi.org/10.2987/moco-31-03-275-277.1 PMID: 26375910

60. Espinosa M, Giamperetti S, Abril M, Seijo A. Vertical transmission of dengue virus in *Aedes aegypti* collected in Puerto Iguazú, Misiones, Argentina. Rev Inst Med Trop Sao Paulo. 2014; 56: 165–167. https://doi.org/10.1590/S0036-46652014000200013 PMID: 24626420

61. Farraudière L, Sonor F, Crico S, Étienne M, Mousson L, Hamel R, et al. First detection of dengue and chikungunya viruses in natural populations of *Aedes aegypti* in Martinique during the 2013–2015 concomitant outbreak. Rev Panam Salud Publica Pan Am J Public Health. 2017; 41: e63.

62. Martínez NE, Dzul-Manzanilla F, Gutiérrez-Castro C, Ibarra-López J, Bibiano-Marín W, López-Damián L, et al. Natural vertical transmission of dengue-1 virus in *Aedes aegypti* populations in Acapulco, Mexico. J Am Mosq Control Assoc. 2014; 30: 143–146. https://doi.org/10.2987/14-6402.1 PMID: 25102601

63. Adelman ZN, Anderson MAE, Wiley MR, Murreddu MG, Samuel GH, Morazzani EM, et al. Cooler temperatures destabilize RNA interference and increase susceptibility of disease vector mosquitoes to viral infection. PLoS Negl Trop Dis. 2013; 7: e2239. https://doi.org/10.1371/journal.pntd.0002239 PMID: 23738025

64. Alto BW, Wiggins K, Eastmond B, Ortiz S, Zirbel K, Lounibos LP. Diurnal temperature range and chikungunya virus infection in invasive mosquito vectors. J Med Entomol. 2018; 55: 217–224. https://doi.org/10.1093/jme/tjx182 PMID: 29040730

65. Dong S, Balaraman V, Kantor AM, Lin J, Grant DG, Held NL, et al. Chikungunya virus dissemination from the midgut of *Aedes aegypti* is associated with temporal basal lamina degradation during blood-meal digestion. PLoS Negl Trop Dis. 2017; 11: e0005976. https://doi.org/10.1371/journal.pntd.0005976 PMID: 28961239

66. Ledermann JP, Borland EM, Powers AM. Minimum infectious dose for chikungunya virus in *Aedes aegypti* and *Ae. albopictus* mosquitoes. Rev Panam Salud Publica Pan Am J Public Health. 2017; 41: e65.

67. Vega-Rúa A, Lourenço-de-Oliveira R, Mousson L, Vazeille M, Fuchs S, Yébakima A, et al. Chikungunya virus transmission potential by local *Aedes* mosquitoes in the Americas and Europe. PLoS Negl Trop Dis. 2015; 9: e0003780. https://doi.org/10.1371/journal.pntd.0003780 PMID: 25993633

68. Vega-Rúa A, Zouache K, Girod R, Failloux A-B, Lourenço-de-Oliveira R. High level of vector competence of *Aedes aegypti* and *Aedes albopictus* from ten American countries as a crucial factor in the spread of chikungunya virus. J Virol. 2014; 88: 6294–6306. https://doi.org/10.1128/JVI.00370-14 PMID: 24672026

69. Alto BW, Wiggins K, Eastmond B, Velez D, Lounibos LP, Lord CC. Transmission risk of two chikungunya lineages by invasive mosquito vectors from Florida and the Dominican Republic. PLoS Negl Trop Dis. 2017; 11: e0005724. https://doi.org/10.1371/journal.pntd.0005724 PMID: 28749964

70. Lourenço-de-Oliveira R, Failloux A-B. High risk for chikungunya virus to initiate an enzootic sylvatic cycle in the tropical Americas. PLoS Negl Trop Dis. 2017; 11: e0005698. https://doi.org/10.1371/journal.pntd.0005698 PMID: 28662031

71. Cigarroa-Toledo N, Blitvich BJ, Cetina-Trejo RC, Talavera-Aguilar LG, Baak-Baak CM, Torres-Chablé OM, et al. Chikungunya virus in febrile humans and *Aedes aegypti* mosquitoes, Yucatan, Mexico. Emerg Infect Dis. 2016; 22: 1804–1807. https://doi.org/10.3201/eid2210.152087 PMID: 27347760

72. Díaz-González EE, Kautz TF, Dorantes-Delgado A, Malo-García IR, Laguna-Aguilar M, Langsjoen RM, et al. First report of *Aedes aegypti* transmission of chikungunya virus in the Americas. Am J Trop Med Hyg. 2015; 93: 1325–1329. https://doi.org/10.4269/ajtmh.15-0450 PMID: 26416113

73. Mordecai EA, Cohen JM, Evans MV, Gudapati P, Johnson LR, Lippi CA, et al. Detecting the impact of temperature on transmission of Zika, dengue, and chikungunya using mechanistic models. PLoS Negl Trop Dis. 2017; 11: e0005568. https://doi.org/10.1371/journal.pntd.0005568 PMID: 28448507

74. Kang DS, Alcalay Y, Lovin DD, Cunningham JM, Eng MW, Chadee DD, et al. Larval stress alters dengue virus susceptibility in *Aedes aegypti* (L.) adult females. Acta Trop. 2017; 174: 97–101. https://doi.org/10.1016/j.actatropica.2017.06.018 PMID: 28648790

75. Maciel-de-Freitas R, Sylvestre G, Gandini M, Koella JC. The influence of dengue virus serotype-2 infection on *Aedes aegypti* (Diptera: Culicidae) motivation and avidity to blood feed. PloS One. 2013; 8: e65252. https://doi.org/10.1371/journal.pone.0065252 PMID: 23755202

76. Sylvestre G, Gandini M, Maciel-de-Freitas R. Age-dependent effects of oral infection with dengue virus on *Aedes aegypti* (Diptera: Culicidae) feeding behavior, survival, oviposition success and fecundity. PloS One. 2013; 8: e59933. https://doi.org/10.1371/journal.pone.0059933 PMID: 23555838

77. Cecílio SG, Júnior WFS, Tótola AH, de Brito Magalhães CL, Ferreira JMS, de Magalhães JC. Dengue virus detection in *Aedes aegypti* larvae from southeastern Brazil. J Vector Ecol J Soc Vector Ecol. 2015; 40: 71–74. https://doi.org/10.1111/jvec.12134 PMID: 26047186

78. Calderón-Arguedas O, Troyo A, Moreira-Soto RD, Marín R, Taylor L. Dengue viruses in *Aedes albopictus* Skuse from a pineapple plantation in Costa Rica. J Vector Ecol J Soc Vector Ecol. 2015; 40: 184–186. https://doi.org/10.1111/jvec.12149 PMID: 26047200

79. Pérez-Castro R, Castellanos JE, Olano VA, Matiz MI, Jaramillo JF, Vargas SL, et al. Detection of all four dengue serotypes in *Aedes aegypti* female mosquitoes collected in a rural area in Colombia. Mem Inst Oswaldo Cruz. 2016; 111: 233–240. https://doi.org/10.1590/0074-02760150363 PMID: 27074252

80. Buckner EA, Alto BW, Lounibos LP. Vertical transmission of Key West dengue-1 virus by *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae) mosquitoes from Florida. J Med Entomol. 2013; 50: 1291–1297. https://doi.org/10.1603/me13047 PMID: 24843934

81. Thangamani S. Vertical transmission of Zika virus in *Aedes aegypti* mosquitoes. Am J Trop Med Hyg. 2016; 95: 1169–1173. https://doi.org/10.4269/ajtmh.16-0448 PMID: 27573623

82. Marini G, Guzzetta G, Rosà R, Merler S. First outbreak of Zika virus in the continental United States: a modelling analysis. Euro Surveill Bull Eur Sur Mal Transm Eur Commun Dis Bull. 2017; 22. https://doi.org/10.2807/1560-7917.ES.2017.22.37.30612 PMID: 28933344

83. Majumder MS, Santillana M, Mekaru SR, McGinnis DP, Khan K, Brownstein JS. Utilizing nontraditional data sources for near real-time estimation of transmission dynamics during the 2015–2016 Colombian Zika virus disease outbreak. JMIR Public Health Surveill. 2016; 2: e30. https://doi.org/10.2196/publichealth.5814 PMID: 27251981

84. Villela D a. M, Bastos LS, DE Carvalho LM, Cruz OG, Gomes MFC, Durovni B, et al. Zika in Rio de Janeiro: assessment of basic reproduction number and comparison with dengue outbreaks. Epidemiol Infect. 2017; 145: 1649–1657. https://doi.org/10.1017/S0950268817000358 PMID: 28240195

85. Ospina J, Hincapie-Palacio D, Ochoa J, Molina A, Rúa G, Pájaro D, et al. Stratifying the potential local transmission of Zika in municipalities of Antioquia, Colombia. Trop Med Int Health TM IH. 2017; 22: 1249–1265. https://doi.org/10.1111/tmi.12924 PMID: 28686321

86. Rojas D, Dean N, Yang Y, Kenah E, Quintero J, Tomasi S, et al. The epidemiology and transmissibility of Zika virus in Girardot and San Andres island, Colombia, September 2015 to January 2016. Euro Surveill. 2016;21. https://doi.org/10.2807/1560-7917.ES.2016.21.28.30283 PMID: 27452806

87.   Fitzgibbon WE, Morgan JJ, Webb GF. An outbreak vector-host epidemic model with spatial structure: the 2015–2016 Zika outbreak in Rio De Janeiro. Theor Biol Med Model. 2017; 14: 7. https://doi.org/10.1186/s12976-017-0051-z PMID: 28347332

88.   Reiner RC, Stoddard ST, Scott TW. Socially structured human movement shapes dengue transmission despite the diffusive effect of mosquito dispersal. Epidemics. 2014; 6: 30–36. https://doi.org/10.1016/j.epidem.2013.12.003 PMID: 24593919

89.   Siraj AS, Oidtman RJ, Huber JH, Kraemer MUG, Brady OJ, Johansson MA, et al. Temperature modulates dengue virus epidemic growth rates through its effects on reproduction numbers and generation intervals. PLoS Negl Trop Dis. 2017; 11: e0005797. https://doi.org/10.1371/journal.pntd.0005797 PMID: 28723920

90.   Yang HM, Boldrini JL, Fassoni AC, Freitas LFS, Gomez MC, de Lima KKB, et al. Fitting the incidence data from the city of Campinas, Brazil, based on dengue transmission modellings considering time-dependent entomological parameters. PloS One. 2016; 11: e0152186. https://doi.org/10.1371/journal.pone.0152186 PMID: 27010654

91.   Yang HM. The transovarial transmission in the dynamics of dengue infection: epidemiological implications and thresholds. Math Biosci. 2017; 286: 1–15. https://doi.org/10.1016/j.mbs.2017.01.006 PMID: 28131789

92.   Campbell KM, Haldeman K, Lehnig C, Munayco CV, Halsey ES, Laguna-Torres VA, et al. Weather regulates location, timing, and intensity of dengue virus transmission between humans and mosquitoes. PLoS Negl Trop Dis. 2015; 9: e0003957. https://doi.org/10.1371/journal.pntd.0003957 PMID: 26222979

93.   Masud MA, Kim BN, Kim Y. Optimal control problems of mosquito-borne disease subject to changes in feeding behavior of *Aedes* mosquitoes. Biosystems. 2017; 156–157: 23–39. https://doi.org/10.1016/j.biosystems.2017.03.005 PMID: 28385591

94.   Lee J-S, Carabali M, Lim JK, Herrera VM, Park I-Y, Villar L, et al. Early warning signal for dengue outbreaks and identification of high risk areas for dengue fever in Colombia using climate and non-climate datasets. BMC Infect Dis. 2017; 17: 480. https://doi.org/10.1186/s12879-017-2577-4 PMID: 28693483

95.   Ernst KC, Walker KR, Reyes-Castro P, Joy TK, Castro-Luque AL, Diaz-Caravantes RE, et al. *Aedes aegypti* (Diptera: Culicidae) longevity and differential emergence of dengue fever in two cities in Sonora, Mexico. J Med Entomol. 2017; 54: 204–211. https://doi.org/10.1093/jme/tjw141 PMID: 28082648

96.   Liu-Helmersson J, Stenlund H, Wilder-Smith A, Rocklöv J. Vectorial capacity of *Aedes aegypti*: effects of temperature and implications for global dengue epidemic potential. PloS One. 2014; 9: e89783. https://doi.org/10.1371/journal.pone.0089783 PMID: 24603439

97.   Velasquez-Castro J, Anzo-Hernandez A, Bonilla-Capilla B, Soto-Bajo M, Fraguela-Collar A. Vector-borne disease risk indexes in spatially structured populations. PLoS Negl Trop Dis. 2018; 12. https://doi.org/10.1371/journal.pntd.0006234 PMID: 29432455

98.   Taber ED, Hutchinson ML, Smithwick EAH, Blanford JI. A decade of colonization: the spread of the Asian tiger mosquito in Pennsylvania and implications for disease risk. J Vector Ecol J Soc Vector Ecol. 2017; 42: 3–12. https://doi.org/10.1111/jvec.12234 PMID: 28504449

99.   Carbajo AE, Vezzani D. Waiting for chikungunya fever in Argentina: spatio-temporal risk maps. Mem Inst Oswaldo Cruz. 2015; 110: 259–262. https://doi.org/10.1590/0074-02760150005 PMID: 25946252

100. Manore CA, Hickmann KS, Xu S, Wearing HJ, Hyman JM. Comparing dengue and chikungunya emergence and endemic transmission in *A. aegypti* and *A. albopictus*. J Theor Biol. 2014; 356: 174–191. https://doi.org/10.1016/j.jtbi.2014.04.033 PMID: 24801860

101. Quiner CA, Parameswaran P, Ciota AT, Ehrbar DJ, Dodson BL, Schlesinger S, et al. Increased replicative fitness of a dengue virus 2 clade in native mosquitoes: potential contribution to a clade replacement event in Nicaragua. J Virol. 2014; 88: 13125–13134. https://doi.org/10.1128/JVI.01822-14 PMID: 25187539

102. Quintero-Gil DC, Ospina M, Osorio-Benitez JE, Martinez-Gutierrez M. Differential replication of dengue virus serotypes 2 and 3 in coinfections of C6/36 cells and *Aedes aegypti* mosquitoes. J Infect Dev Ctries. 2014; 8: 876–884. https://doi.org/10.3855/jidc.3978 PMID: 25022298

103. Serrato-Salas J, Izquierdo-Sánchez J, Argüello M, Conde R, Alvarado-Delgado A, Lanz-Mendoza H. *Aedes aegypti* antiviral adaptive response against DENV-2. Dev Comp Immunol. 2018; 84: 28–36. https://doi.org/10.1016/j.dci.2018.01.022 PMID: 29408269

104. Vazeille M, Gaborit P, Mousson L, Girod R, Failloux A-B. Competitive advantage of a dengue 4 virus when co-infecting the mosquito *Aedes aegypti* with a dengue 1 virus. BMC Infect Dis. 2016; 16: 318. https://doi.org/10.1186/s12879-016-1666-0 PMID: 27390932

105. Cromwell EA, Stoddard ST, Barker CM, Van Rie A, Messer WB, Meshnick SR, et al. The relationship between entomological indicators of *Aedes aegypti* abundance and dengue virus infection. PLoS Negl Trop Dis. 2017; 11: e0005429. https://doi.org/10.1371/journal.pntd.0005429 PMID: 28333938

106. Lana RM, Gomes MF da C, Lima TFM de, Honório NA, Codeço CT. The introduction of dengue follows transportation infrastructure changes in the state of Acre, Brazil: A network-based analysis. PLoS Negl Trop Dis. 2017; 11: e0006070. https://doi.org/10.1371/journal.pntd.0006070 PMID: 29149175

107. Eisen L, García-Rejón JE, Gómez-Carro S, Nájera Vázquez M del R, Keefe TJ, Beaty BJ, et al. Temporal correlations between mosquito-based dengue virus surveillance measures or indoor mosquito abundance and dengue case numbers in Mérida City, México. J Med Entomol. 2014; 51: 885–890. https://doi.org/10.1603/me14008 PMID: 25118425

108. Liebman KA, Stoddard ST, Reiner RC, Perkins TA, Astete H, Sihuincha M, et al. Determinants of heterogeneous blood feeding patterns by *Aedes aegypti* in Iquitos, Peru. PLoS Negl Trop Dis. 2014; 8: e2702. https://doi.org/10.1371/journal.pntd.0002702 PMID: 24551262

109. Oliveira R de MAB, Araújo FM de C, Cavalcanti LP de G. Entomological and epidemiological aspects of dengue epidemics in Fortaleza, Ceará, Brazil, 2001–2012. Epidemiol E Serv Saude Rev Sist Unico Saude Bras. 2018; 27: e201704414. https://doi.org/10.5123/s1679-49742018000100014 PMID: 29451615

110. Monaghan AJ, Morin CW, Steinhoff DF, Wilhelmi O, Hayden M, Quattrochi DA, et al. On the seasonal occurrence and abundance of the Zika virus vector mosquito *Aedes aegypti* in the contiguous United States. PLoS Curr. 2016; 8. https://doi.org/10.1371/currents.outbreaks.50dfc7f46798675fc63e7d7da563da76 PMID: 27066299

111. Lo D, Park B. Modeling the spread of the Zika virus using topological data analysis. PloS One. 2018; 13: e0192120. https://doi.org/10.1371/journal.pone.0192120 PMID: 29438377

112. da Cruz Ferreira DA, Degener CM, de Almeida Marques-Toledo C, Bendati MM, Fetzer LO, Teixeira CP, et al. Meteorological variables and mosquito monitoring are good predictors for infestation trends of *Aedes aegypti*, the vector of dengue, chikungunya and Zika. Parasit Vectors. 2017; 10: 78. https://doi.org/10.1186/s13071-017-2025-8 PMID: 28193291

113. Stewart-Ibarra AM, Lowe R. Climate and non-climate drivers of dengue epidemics in southern coastal ecuador. Am J Trop Med Hyg. 2013; 88: 971–981. https://doi.org/10.4269/ajtmh.12-0478 PMID: 23478584

114. da Rocha Taranto MF, Pessanha JEM, dos Santos M, dos Santos Pereira Andrade AC, Camargos VN, Alves SN, et al. Dengue outbreaks in Divinopolis, south-eastern Brazil and the geographic and climatic distribution of *Aedes albopictus* and *Aedes aegypti* in 2011–2012. Trop Med Int Health TM IH. 2015; 20: 77–88. https://doi.org/10.1111/tmi.12402 PMID: 25328988

115. Souza-Neto JA, Powell JR, Bonizzoni M. *Aedes aegypti* vector competence studies: a review. Infect Genet Evol J Mol Epidemiol Evol Genet Infect Dis. 2019; 67: 191–209. https://doi.org/10.1016/j.meegid.2018.11.009 PMID: 30465912

116. Lourenço-de-Oliveira R, Marques JT, Sreenu VB, Atyame Nten C, Aguiar ERGR, Varjak M, et al. *Culex quinquefasciatus* mosquitoes do not support replication of Zika virus. J Gen Virol. 2018; 99: 258–264. https://doi.org/10.1099/jgv.0.000949 PMID: 29076805